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Reply: Can Silent Brain Lesions Be a Target to Guide Anticoagulation Treatment in Patients With Low-Risk Atrial Fibrillation to Reduce Cognitive Impairment?

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Response to “ Can be Silent Brain Lesions a Target to Guide Anticoagulation Treatment in Patients with Low-Risk Atrial Fibrillation to Reduce Cognitive Impairment?” by Ammirati et al.

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As raised by Ammirati et al. (1) the recent evidence (2) that prevalence of silent cerebral ischemia (SCI) in patients with paroxysmal and persistent atrial fibrillation (AF) and controls in sinus rhythm implies several clinically relevant issues.

The finding that approximately half of controls presented at least an area of SCI is strongly hypothesis generating. On the other side the prevalence of other cardio-embolic sources, as patent foramen ovale or nonstenotic carotid plaques material, should not be differently stratified within cases and controls, therefore not biasing main study results.

Indeed the question whether SCI represent a target for oral anticoagulation (OAC) to prevent cognitive impairment is crucial. In fact, risk stratification, to date, relies on clinical scores (CHA2DS2-VASc score) based on symptomatic cerebral events only and, anyway, fallacious.

The occurrence of cerebrovascular events in AF patients, despite low risk score remains unfortunately not unusual (3) warranting introduction of “new” markers, as left atrial appendage morphology (4,5) and specific echocardiographic parameters (6). Limited literature has assessed whether antiaggregants/OAC may prevent/reduce SCI. One small study (7) suggested that aspirin attenuates SCI incidence in AF patients, preventing events also in early stages of AF-related cerebral damage and, consequently, cognitive decline. However, this option is not recommended

due to the detrimental effects of antiaggregants with bleeding (8). We fully agree with Ammirati et al that further prospective randomized trials are needed to evaluate the possible reduction of SCI by OAC. In the meantime aggressive rhythm control strategy is mandatory to prevent clinical and silent cerebral ischemia by sinus rhythm restoration and maintenance.

References

1. Ammirati E, Scotti I and Camici PG. Can be Silent Brain Lesions a Target to Guide Anticoagulation Treatment in Patients with Low-Risk Atrial Fibrillation to Reduce Cognitive Impairment? *J Am Coll Cardiol* 2014 in press.
2. Gaita F, Corsinovi L, Anselmino M et al. Prevalence of silent cerebral ischemia in paroxysmal and persistent atrial fibrillation and correlation with cognitive function. *J Am Coll Cardiol* 2013;62:1990-7.
3. Potpara TS, Polovina MM, Licina MM et al. Reliable identification of "truly low" thromboembolic risk in patients initially diagnosed with "lone" atrial fibrillation: the Belgrade atrial fibrillation study. *Circ Arrhythm Electrophysiol* 2012;2:319-26.
4. Di Biase L, Santangeli P, Anselmino M et al. Does the left atrial appendage morphology correlate with the risk of stroke in patients with AF? Result from a multicenter study. *J Am Coll Cardiol* 2012;60:531–38.
5. Anselmino M, Scaglione M, Di Biase L et al. Left atrial appendage morphology and silent cerebral ischemia in atrial fibrillation patients. *Heart Rhythm* 2014; 11(1):2-7.
6. Tamura H, Watanabe T, Nishiyama S et al. Prognostic value of low left atrial appendage wall velocity in patients with ischemic stroke and atrial fibrillation. *J Am Soc Echocardiogr* 2012; 5:576-83.
7. Sato H, Koretsune Y, Fukunami M, et al. Aspirin attenuates the incidence of silent brain lesions in patients with non-valvular atrial fibrillation. *Circ J* 2004;68:410–16.
8. Olesen JB, Lip GY, Lindhardsen J et al. Risks of thromboembolism and bleeding with thromboprophylaxis in patients with atrial fibrillation: A net clinical benefit analysis using a 'real world' nationwide cohort study. *Thromb Haemost* 2011;4:739-49.